A CLINICAL STUDY OF ACUTE DEEP VEIN THROMBOSIS

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CERTIFICATE

This is to certify that this dissertation entitled **A clinical study of Acute Deep Vein Thrombosis** submitted by **Dr.M.Bhuvaneswaran** appearing for M.Ch Vascular Surgery Examination, February 2006, is a bonafide record of work done by him under my direct guidance and supervision.

I forward this to The Tamilnadu Dr.MGR Medical University, Chennai.

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CONTENTS

INTRODUCTION

Venous thromboembolism is responsible for considerable morbidity and mortality both in hospital and in the community. The impact of venous thromboembolism on morbidity and mortality is largely unrecognised in daily practice because of low autopsy rates. Despite thromboembolic prophylaxis, symptomatic and asymptomatic pulmonary embolism occurs in approximately 25% of surgical patients and is responsible for 3% of surgical inpatient deaths. Approximately 25% of patients who have a fatal pulmonary embolism have had recent surgery. An understanding of the underlying epidemiology, pathophysiology and natural history of deep vein thrombosis is essential in guiding appropriate prophylaxis, diagnosis and treatment. Recognition of the underlying risk factors and an appreciation of the multifactorial nature of deep vein thrombosis may facilitate the identification of situations likely to provoke thrombosis in high-risk individuals as well as the further evaluation of those with an unexplained thromboembolism. An understanding of the natural history of deep vein thrombosis is similarly important in defining the relative risk and benefits of anticoagulation as well as the duration of treatment in individual patients.

AIM

To Assess

i) Mode of presentation.

ii) Pattern of limb and deep vein involvement.

iii) Common predisposing factors.

MATERIALS AND METHODS

In this study 119 cases of deep vein thrombosis admitted in vascular surgery ward and other wards of Government General Hospital, Chennai between July 2004 and July 2005 were analysed.

All patients underwent a routine clinical examination, serial limb girth measurement and duplex scan of the deep veins for confirmation of the diagnosis chest X ray, ECG, ECHO were done to detect pulmonary embolism. Tests for evaluating the presence of hypercoagulable states were done in a few patients who could afford.

A structured proforma for clinical assessment, diagnosis and management was framed and the data compiled and analysed.

All patients were treated by lower limb elevation, absolute bed rest for one week, intravenous bolus unfractionated heparin for one week, tablet Acitrom 4 mg once daily to be overlapped with heparin for 5 days and once INR was above 2, heparin was stopped and compression bandaging of the lower limb was done and patient made to ambulate. The patients were discharged by the eleventh or twelfth day and reviewed every 2 weeks for the first month with prothrombin time and INR values. Lower limb graduated compression stockings were provided. Patients were maintained an oral anticoagulants for a period of 3 to 6 months.

REVIEW OF LITERATURE

The first report on deep vein thrombosis was published in 1810, Ferriar described a patient with phlegmasia alba dolens. In 1859 Virchow described his revolutionary discovery of the three main causes of deep vein thrombosis.

EPIDEMIOLOGY

Deep vein thrombosis most often complicates the course of severally ill, hospitalised patients but may also affect ambulatory and otherwise healthy subjects (1). Even if the importance of venous thromboembolic disorders as a major cause of morbidity and mortality has been gaining attention in the past two decades, the true incidence of venous thromboemoblism in the general population remains difficult to determine. This is because of the clinically silent nature of most thromboses as well as the non specific signs and symptoms as discussed by Barnes RW et al. 1975 (3) and Cranley JJ et al. 1976 (16).

PATHOPHYSIOLOGY

In 1859, Virchow postulated the triad in the development of venous thrombosis

(i) stasis

(ii) abnormalities of blood,

(iii) endothelial injury.

Mechanical venous injury clearly plays a role in thrombosis associated with direct venous trauma (Dennis JW. 1993) (17). However, focal venous injury cannot account for bilateral venous thromboses in

77%, compared to unilateral 23%. Overt endothelial injury is neither necessary nor sufficient to cause thrombosis. The potential role of biochemical injury to the venous endothelium has been explained by Mammen EF (1992) (43).

Regardless of etiology, most venous thrombi originate in soleal veins or behind valve pockets according to Sevitt. S. (1974) (55). Stasis causes hypoxia at the depths of valve cusps and may induce endothelial injury. Furthermore stasis allows accumulation of activated coagulation factors and consumption of inhibitors (Thomas SP et al 1983) (60). But stasis in isolation appears to be inadequate stimulus for thrombosis.

Activation of coagulation appears to be of critical importance in the pathogenesis of deep vein thrombosis. Increased levels of these markers have been described in association with surgery (Kambayashi J. et al. 1990), oral contraceptives (Vonkulla et al 1971) and malignancy (Falunga. A et al. 1994). Deep vein thrombosis thus appears to be a multifactorial phenomenon, with convergence of several pathologic factors.

RISK FACTORS

Approximately 47% of patients with a document deep vein thrombosis have one or more recognised risk factors with the incidence of venous thromboembolism increasing with the number of risk factors

(Anderson FA et al. 1992) (2).

OLD AGE

In the Framingham study, advanced age was significantly associated with fatal pulmonary embolism (Goldhaber SZ et al. 1983) (25). In their study of out of hospital patients, Anderson et al. reported an association between advancing age and thrombosis. In virtually all studies recruiting consecutive patients with documented venous thromboembolism for scientific purpose, old patients formed the large majority of the entire cohort.

PROLONGED IMMOBILISATION

The association between lack of mobility and venous thrombosis has been confirmed in a number of autopsy and clinical studies. The contribution of immobilisation as an independent risk factor for venous thromboemoblism emerged in a study in hemiplegic patients, where the non paralysed leg served as control. Waslow et al (1972) (62) found evidence of deep vein thrombosis using the radiofibrinogen I^{125} scanning technique, in 60% of paralysed limbs compared with only 7% in the non paralysed limbs.

SURGERY

Patients who undergo prolonged surgery are at risk of developing deep vein thrombosis and this risk continuous after discharge from

hospital. Surgery constitutes a spectrum of risk that is influenced by patient age, coexistent thrombotic risk factors, type of procedure, extent of surgical trauma length of procedure and duration of postoperative immobilization.

Clagett GP et al (1995) (9) showed the incidence of deep vein thrombosis to be 19% after general surgery, 24% following neurosurgery, 50% for hip and knee arthroplasties. Macklan NS et al (1996) showed that 51% of gynaecologic patients developed deep vein thrombosis following initial discharge. Surgery in accompanied by perioperative immobilization, transient hypercoagulable state and decreased fibrinolytic state (Kambayashi J. et al. 1990) (35).

MAJOR TRAUMA

The frequency of venous thromboembolic complications after major trauma has been recently investigated with systematic phlebography by Geerts W. et al (1994) (21) in patients admitted to a regional trauma unit. They found that although most patients were asymptomatic, 58% of them had deep vein thrombosis. Other associated risk factors as reported by Napolitano LM et al (1995) (45) were a hospital stay of greater than 7 days, increased Injury Severity Score ISS and the duration of immobilisation.

MALIGNANT DISEASES

After the initial observation by Armond Trousseau in 1865, numerous studies have addressed the relationship between cancer and thrombosis. Postmortem studies

conducted by Sproul EE (1983) (59) have demonstrated an increased incidence of deep vein thrombosis in patients who died of cancer, particularly those with mucinous carcinomas of the pancreas, lung and gastrointestinal tract.

The conditions that make cancer patients susceptible to thrombosis are the following: prolonged immobilisation, surgical intervention, chemotherapy and the presence of central venous catheters. Cancer patients receiving immunosuppressive or cytotoxic chemotherapy are at a higher risk for venous thrombosis (Levine MN et al 1988) (39) Liebman HA et al (1982) showed that chemotherapy with L-asparaginase is a risk factor for deep vein thrombosis owing to reduced plasma antithrombin activity.

CENTRAL VENOUS CATHETERS

A population based case control study by Hert JA et al (2000) (29) showed a six fold increased risk of deep vein thrombosis among patients with central venous catheter or a transvenous pacemaker.

PREGNANCY AND PUERPERIUM

Population based studies have suggested that deep vein thrombosis complicates 0.1% to 0.7% of pregnancies. Deep vein thrombosis in pregnancy has been attributed to (i) venous outflow obstruction due to uterine compression (Toglia MR et al. 1996) (61). (ii) Decreased protein S levels (iii) Increased coagulation factors (Beller EK et al 1982) (6).

Oral Contraceptives and Hormone replacement therapy

Koster et al (37) preformed a meta analysis of controlled studies (1993) which reveals three fold increased risk of deep vein thrombosis in oral contraceptive pill users.

Gertsman BB (1991) (22) proved the dose response between the content of estrogen and the risk for deep vein thrombosis.

Grady B et al (1997) (26) showed that hormone replacement therapy is associated with two to four fold increased risk of deep vein thrombosis.

PREVIOUS VENOUS THROMBOEMBOLISM

In a case control study Coon Willis (14) found that the risk of recurrent deep vein thrombosis after an initial episode was higher at all stages of follow up for patients with a history of venous thromboembolism compared to those without a previous episode.

PRIMARY HYPERCOAGULABLE STATES

40-45% of patients with deep vein thrombosis can be characterised as thrombophilic.

Protein C, Protein S and Antithrombin III deficiencies may be associated with 5 to 10% of thrombotic events. Patients with heterozygous deficiency states often present with a first thrombotic event before 40 years of age, approximately 50% of such events being related to predisposing situations (Girolami A et al. 1995) (24).

PROTEIN C DEFICIENCY

Protein C is a vitamin K dependent glycoprotein synthesized by the liver. During coagulation, protein C is activated slowly by thrombin. This reaction is markedly accelerated by the binding of thrombin with thrombomodulin. The high affinity binding of thrombin to thrombomodulin produces a 20,000 fold increase in the rate of protein C activation.

In the general population symptomatic protein C deficiency has been estimated to be between 1:16,000 and 1:36,000 and that of asymptomatic protein C deficiency between 1:200 and 1:300. Recent studies indicate that protein C deficiency can be expressed clinically as either an autosomal dominant or an autosomal recessive trait. The deficiency of protein C appears to be identical in both forms of deficiency, but for obscure reasons, their clinical manifestations are different.

When protein C deficiency is expressed as an autosomal dominant trait, over 70% of affected subjects will develop venous thromboembolism during their life time. In contrast, when protein C deficiency is expressed clinically as an autosomal recessive trait, venous thrombosis is extremely uncommon, unless at occurs in the homozygous or double heterozygous state.

PROTEIN S DEFICIENCY

The principal cofactor of activated protein C is protein S a vitamin K dependent glycoprotein, produced by the liver and endothelial cells. It circulates in plasma as a free form (40%) and as a noncovalent complex with C4b-binding protein. Protein S acts by enhancing the affinity of activated protein C for negatively charged phospholipids, forming a membrane bound antiphospholipids – protein S complex that renders factors Va and VIIIa more easily accessible to activated protein C mediated cleavage.

The frequency of protein S deficiency in the general population is unknown. In unselected and selected patients, presenting with the first episode of venous thrombosis, the frequency averages 2.2% and 3% respectively.

ACTIVATED PROTEIN C RESISTANCE

This is the most common cause of inherited thrombosis in Western population. In more than 90% of cases, it is caused by a single point mutation in the factor V gene. This results in the substitution of arginine by glutamine at position 506 of the polypeptide chain of factor V. This prevents cleavage of factor Va at this site by activated protein C and hence delays the inactivation of factor Va. There is a surprisingly high prevalence 3 to 10% among caucasians.

The penetrance of thrombosis in activated protein C resistant individuals is highly variable. Some individuals never develop thrombosis, whereas others develop recurrent, severe thrombotic events at a young age.

Activated protein C resistance (factor v Leiden) in present is 3 to 10% of caucasians and virtually absent in other races (Zoller B 1997) (66).

Rees MM et al (1943) (51) elaborated on hyperhomocysteinemia as a result of homozygous cystathionine ß-synthase deficiency and these patients develop homocystinuria, premature vascular disease and thrombosis.

ANTIPHOSPHOLIPID ANTIBODY SYNDROME

Antiphospholipid antibodies are a heterogeneous group of immunoglobulins directed against negatively charged phospholipids, protein-phospholipid complexes, or plasma proteins such as β-2 glycoprotein. Antiphospholipid antibody includes anticardiolipin and lupus anticoagulant antibodies.

Both lupus anticoagulant and anticardiolipin antibodies are characteristically found in patients with systemic lupus erythematosus, but can be observed in other

autoimmune diseases and even in the absence of any underlying disease. The presence of these antibodies has been associated with the clinical features of the so-called "Antiphospholipid Antibody syndrome" (APS), including arterial and venous thrombosis, recurrent fetal loss and thrombocytopenia.

Antiphospholipid antibodies have been estimated to occur in 2% of the general population. Their frequency in systemic lupus erythematosus varies between 30 and 40%. The true clinical relevance of antiphospholipid antibodies in pattern suffering an episode of venous thromboembolism is still unknown.

ANTITHROMBIN III DEFICIENCY

Antithrombin III is a single chain glycoprotein, synthesised by the liver. It is the primary inhibitor of thrombin and also inhibits other serine proteases involved in the intrinsic clotting system ie. factor Xa, IXa, XIa XIIa and kallikrein. Protease inactivation by antithrombin involves the formation of a complex between the active site of the protease and the reactive centre of antithrombin.

Inhibition of most of the blood coagulation proteinases is relatively slow but is markedly accelerated by the binding of heparin to antithrombin. In unselected patients presenting with the first episode of venous thromboembolism, the frequency is approximately 1% and 2.5% in selected patients. Although it is difficult to obtain exact figures on the risk of thrombosis in affected persons, review of the literature indicates that more than 50% of affected persons develop thromboembolic events before the age of 50 years. In most cases, patients develop deep vein thrombosis and pulmonary embolism, there are also occassional reports of thrombosis in unusual sites and in the arterial circulation. Antithrombin III deficiency confers a higher risk of thrombosis than deficiencies of protein C and protein S. This is confirmed by the fifty fold difference

in the prevalence among patients with a first event of deep vein thrombosis and the prevalence in a healthy population.

CLINICAL ASSESSMENT

Wells and associates (1995) (63) demonstrated that combining the use of a model of clinical probability of deep vein thrombosis with ultrasound examination decreased the false positive or false negative diagnoses. They found that patients with a high clinical suspicion of deep vein thrombosis had a 85% chance and having a phlebographically proven deep vein thrombosis.

DIAGNOSTIC TECHNIQUES

The presently available techniques for the objective diagnosis of deep vein thrombosis include invasive and non invasive methods and biochemical assays.

VENOGRAPHY

Contrast venography is considered as gold standard for the diagnosis of deep vein thrombosis. The diagnosis of lower extremity deep vein thrombosis was largely a clinical one until the 1960s, when contrast venography became a part of standard medical practice. Lower extremity venography was used by dos Santas in 1938 to confirm a clinically suspected diagnosis of deep vein thrombosis.

In 1940, Bauer published details of normal venographic anatomy and of the venographic appearance of acute and chronic deep vein thrombosis. Diagnostic criteria for deep vein thrombosis were established in 1963 by De Weese and Rogoff. In the past decades, the technique used for lower extremity ascending venography has been modified many times. However even as the technique has been refined and the

examination has been made safer and less painful with the use of modern contrast agents, it remains a relatively invasive study that requires an often painful venipuncture, injection of iodine based contrast agents and exposure of patient to ionising radiation. Comerota AJ et al (1985) (13) showed that this test may not be feasible or is inadequate for interpretation in upto 20% of patients.

Because of the numerous disadvantages and the better results of duplex scanning, venography is infrequently used today.

INDIRECT PHYSIOLOGIC TESTS

VENOUS PLETHYSMOGRAPHY

Plethysmography is a noninvasive method of detecting blood volume changes in an extremity. It is dependent an hemodynamic principles of volume displacement and venous capacitance. The techniques strain gauge plethysmography, impedance plethysmography, segmental air plethysmography and photoplethysmography are all based on the same principle.

With the patient in the supine position, lower extremity veins are not completely filled and are able to accommodate increased blood volume before reaching maximal capacitance. With normal venous outflow, positional change or release of an externally inflated cuff will result in rapid emptying of the leg veins. In the presence of venous obstruction, the peripheral venous pressure is already elevated with an increased baseline venous volume and maximal venous capacitance is reached with smaller volume changes.

Comerato and associates reported a sensitivity of 83% proximal deep vein thrombosis in patients after clinical diagnosis in contrast to only 33% for surveillance of

high risk patients. This difference can be attributed to the increased presence of non occlusive thrombi and calf vein thrombosis in asymptomatic patients undergoing post operative surveillance.

Sequential studies may improve accuracy with increased venous occlusion from propagation of thrombus into proximal veins. Sequential studies can also be used to quantify resolution of thrombus in patients whose abnormal impedance plethysmography result has become normal. Wheeler HB et al (65) shared that impedance plethysmography was reasonably reliable in detecting proximal deep vein thrombosis, but were unreliable in asymptomatic patients as many of them had non-occlusive thrombus.

PHLEBORHEOGRAPHY

It is a variation of venous plethysmography, it measures phasic volume changes in the lower extremity that occur with normal respiratory cycle. A normal breathing pattern produces a cyclic decrease in lower limb volume with expiration and increase with inspiration, related to the effect on venous outflow of intraabdominal pressure from the rising and falling diaphragm. In the presence of deep venous obstruction, these respiratory waves are absent.

Overall sensitivity for phleborheography in the lower extremity has ranged from 79% to 93% and specificity has ranged from 87% to 97%. Phleborheography is most accurate for proximal deep vein thrombosis in symptomatic patients. It is least sensitive for calf vein thrombosis and in asymptomatic patients. It is technically demanding and calls for more experience in performance and interpretation than other venous plethysmographic techniques. It is also limited by the same sources of error as

other venous plethysmographic tests, including patient cooperation, muscle spasm and extrinsic venous compression.

THERMOGRAPHY

Venous thrombosis produces an inflammatory reaction with release of vasoactive substances, causing a microcirculatory hyperemic response and increase in local skin temperature of up to 2°C. This temperature change may not be evident clinically, but it can be detected with thermographic techniques. A normal scanning thermogram shows an even distribution of temperature throughout the limb with anatomic cool zones noted over the patella and surface of tibia. A gradual temperature gradient is noted moving towards the warmer proximal portion of the extremity. In the presence of deep vein thrombosis, a diffuse area of elevated temperature is observed in the calf or thigh with loss of patellar and tibial cooling.

RADIONUCLIDE TEST

Iodine 125 – Fibrinogen uptake

The detection of thrombus by uptake of radiolabeled fibrinogen was first described by Hobbs and Davies in 1960 and was introduced to clinical use by Kakkar and associates in 1970. The matrix of fresh thrombus is composed largely of fibrin, which is formed from its soluble precursor fibrinogen. The I^{125} fibrinogen update test is performed with intravenous injection of 100 μCi of I^{125} fibrinogen into a peripheral vein, followed by interval measurement along the lower extremity for radiolabeled fibrin incorporation into fresh thrombus.

Validation studies have determined I^{125} fibrinogen update to be a more accurate test for calf vein thrombosis than for proximal deep vein thrombosis. Clinical use is

limited by (i) failure to detect thrombus in the proximal thigh or pelvic veins because of scatter from the bladder or attenuation from the overlying tissue in the upper thigh. (ii) Delay in diagnosis because of the time needed for incorporation of fibrinogen by the thrombus. (iii) Risk of donor blood products.

VENOUS DUPLEX IMAGING

Real time gray scale imaging and Doppler flow are combined in duplex and colour flow instruments. At each site, venous compressibility and the presence of echogenic thrombus are evaluated on the B mode image, while Doppler is used to assess venous flow characteristics. Venous incompressibility is the most widely used diagnostic criterion for acute deep vein thrombosis. Other diagnostic criteria include thrombus visualization, absent or diminished spontaneous venous flow, absence of respiratory phasicity and absent or incomplete color filling of lumen. Adjunctive criteria include increased venous diameter less than 50% diameter increase with valsalva maneuver and immobile valve cusps.

Duplex scan has some limitations. Imaging of the inferior vena cava and iliac veins are precluded by overlying bowel gas and their deep location often prevents assessment of compressibility. Adequate examination of the tibial and peroneal veins may be impeded by large calf size, edema or operator inexperience.

This is the current main stay of the diagnosis of deep vein thrombosis. Comerota AJ et al (12) have reported good results in high risk asymptomatic patients and is more accurate than indirect physiologic tests and hence has replaced them for initial screening of patients (Heijober H et al. 1993) (28).

MAGNETIC RESONANCE VENOGRAPHY

Carpenter AJ et al (1993) (8) demonstrated that MRV has excellent sensitivity for the diagnosis of proximal venous thrombosis in comparison with ascending photography. The true value of MRV is likely to be in the patients with pelvic and venacaval thrombosis for which traditional diagnostic studies are inadequate.

In a study of 101 lower extremity, abdominal and pelvic venous systems, magnetic resonance venography was compared with duplex ultrasound and contrast venography and was found to be nearly perfect in detection of deep vein thrombosis. Magnetic resonance venography compared favourably with duplex scanning of the lower extremities and consistently imaged the pelvic veins and vena cava, which are often inaccessible to duplex scan.

Magnetic resonance imaging / Magnetic resonance venography not only accurately identifies the site of deep vein thrombosis, but also allows the identification of conditions that may cause, mimic or be associated with venous thrombosis, such as ruptured Baker's cyst, cellulitis, muscle tears, hematomas and external venous compression from tumours or other anatomic anomalies. Magnetic resonance imaging and Magnetic resonance venography technology can serve as a useful adjuvant imaging modality that offers a non invasive approach to the difficult diagnostic problem of assessing non-neoplastic venous thrombosis of the renal veins. They can also depict normal and abnormal hepatic and portal venous anatomy is relation to the hepatic parenchyma and thus may be useful in planning transjugular intrahepatic portosystemic stent shunt.

D-DIMER ASSAY

D-dimer, a degradation product resulting from fibrinolysis of complexed fibrin was proved to be useful in evaluating patients with suspected deep vein thrombosis. Although D-dimer levels are elevated in postoperative and acutely ill patients, (Rowbotham BJ et al 1987), Ginsberg colleagues (23) showed that a normal D-dimer value had a negative predictive value of 97%.

It is also elevated in disseminated intravascular coagulation, arterial thrombosis or sickle cell crisis. Most laboratory assays for D-dimer use a monoclonal antibody against epitopes on the D-dimer fragment that are absent on fibrinogen, fibrin or fibrin degradation products.

The ELISA tests use both a monoclonal antibody to D-dimer and a second antibody to either fibrin or fibrinogen to quantitate D-dimer concentrations as low as 30 to 80 ng/mL. Latex agglutination assays use a single monoclonal antibody attached to latex beads to detect D-dimer concentration in the range of 200 to 500 ng/ml but only a few minutes to perform in contrast to the several hours of ELISA. Accuracy improves with increasing D-dimer levels corresponding to increasing extent of deep vein thrombosis on venograms. The clinical role of D-dimer testing, alone or in combination with other diagnostic studies for deep vein thrombosis, has yet to be determined but it is limited principally by its low specificity.

PREVENTION OF DEEP VEIN THROMBOSIS

Without prophylaxis, the frequency of fatal pulmonary embolism ranges from 0.1% to 0.8% in general surgical patients (Skinner DB 1967) (58) 2 to 3% in elective hip

replacement patients (Coventry MB et al 1973) (15) and 4% to 7% in patients undergoing surgery for a fracture hip (Esteland G. et al. 1986).

Salzman EW (53) reported that prevention of deep vein thrombosis and pulmonary embolism is more cost effective than treatment of the complications when they occur.

LOW DOSE HEPARIN

The effectiveness of low dose unfractionated heparin for preventing deep vein thrombosis has been established by multiple randomized clinical trials. Low dose subcutaneous heparin in usually given in a dose of 5000 units 2 hours preoperatively and postoperatively every 8 or 12 hours. Pooled data from meta-analyses confirm that low dose heparin significantly reduces the incidence of all deep vein thrombosis, proximal deep vein thrombosis and all pulmonary emboli including fatal pulmonary emboli (Collings R et al 1988) (10). The International Multicentre trial (32) also established the effectiveness of low dose heparin for preventing fatal pulmonary embolism, a clinically and significantly striking reduction from 0.7% to 0.0%.

ADJUSTED DOSE HEPARIN

The use of adjusted dose subcutaneous heparin has been effective for prophylaxis compared with low dose heparins in patients undergoing total hip replacement, but it has not become popular because of the time and expense required for laboratory monitoring.

LOW MOLECULAR WEIGHT HEPARIN

In randomized clinical trials comparing LMW heparin with unfractionated heparins, the low molecular weight heparins given once daily or twice daily have been shown to be as effective as or more effective than unfractionated heparin in preventing thrombosis (Kakkar VV et al 1993) (34).

These trials also showed low frequencies of bleeding for low molecular weight heparin and low dose unfractionated heparin. Clagett GP et al have shown that LMWH reduces the incidence of postoperative deep vein thrombosis by 80% in general surgery. Prandoni P et al (1996) (50) reported that LMWH at doses twice as high as those recommended for deep vein thrombosis prevention in general surgery, reduces the incidence of postoperative deep vein thrombosis in elective hip surgery by 71%, knee surgery but 41% and by 44% in hip fracture surgery.

ORAL ANTICOAGULANTS

For prophylaxis oral anticoagulants can be commenced preoperatively at the time of surgery, or in the early postoperative period. Oral anticoagulants begun at the time of surgery or in the early postoperative period may not prevent small venous thrombi from forming during or soon after surgery, because the antithrombolic effect is not achieved until the third or fourth postoperative day. However they are effective in inhibiting the extension of these thrombi, there by preventing clinically important venous thromboembolism.

Leclerc JR et al (1996) (38) compared postoperative warfarin with enoxaparin and found no difference in the incidence of postoperative venous thrombosis or bleeding.

INTERMITTENT LEG COMPRESSION

The use of intermittent pneumatic leg compression prevents deep vein thrombosis by enhancing blood flow in the deep veins of the legs thereby preventing venous stasis. It also increases blood fibrinolytic activity which may contribute to its antithrombolic properties. This was found effective in preventing venous thrombosis in moderate risk general surgical patients (Robert VC et al 1972) (52) and in patients undergoing neurosurgery (Skillman JJ et al 1978) (57). This is virtually free of clinically important side effects. These devices should be used for the entire period until the patient is full ambulatory, with only temporary removal for nursing care or physiotherapy.

GRADUATED COMPRESSION STOCKINGS

These are simple, safe and moderately effective form of thrombo prophylaxis. They increase the velocity of venous flood flow. Wells PS et al (64) after a meta analysis concluded that there was a highly significant risk reduction in patients at moderate risk of postoperative thromboembolism.

TREATMENT

The objectives of treatment in patients with venous thromboembolism are (1) to prevent death from pulmonary embolism (2) to prevent recurrent venous thromboembolism and (3) to prevent to post-phlebitic syndrome.

HEPARIN THERAPY

The anticoagulant activity of unfractionated heparin depends upon a unique pentasaccharide that binds to antithrombin III and potentiates the inhibition of thrombin and activated factor x by AT III. In addition heparin catalyses the inactivation of thrombin by another plasma cofactor II. Heparin has a number of other effects such as

i) release of tissue factor pathways

ii) binding to numerous plasma and platelet proteins, endothelial cells and leukocytes.

iii) suppression of platelet function

iv) increase in vascular permeability

The anticoagulant response to a standard dose of heparin varies widely among patients. This makes it necessary to monitor activated partial thromboplastin time and to titrate the dose.

The accepted anticoagulant therapy for venous thromboembolism is a combination of continuous intravenous heparin therapy and oral warfarin. The simultaneous use of heparin and warfarin with heparin for initial 5 days only has become clinical practice for all patients with venous thromboembolism as per Gallus et al (1986) (20).

Hull et al recommended heparin infusion therapy, where aPTT was maintained 1.5 times the control. Data from these trials indicate that failure to achieve the

therapeutic aPTT within 24 hours was associated with 23% subsequent recurrent venous thromboembolism rate. Hull et al (1997) (31) also reported that bleeding during heparin therapy is more closely related to underlying clinical risk factors than to aPTT elevation above therapeutic range.

LMW heparin differ from unfractionated heparin in numerous ways. According to Fareed J et al (1997) (19), there is increased bioavailability, prolonged half life and predictable antithrombotic response permitting treatment without monitoring.

Prandoni P. et al (1992) (50) compared subcutaneous unmonitored LMW heparin with continuous intravenous heparin for the treatment of proximal venous thrombosis and found that LMW heparin was as effective and safe as unfractionated heparin.

COMPLICATIONS OF HEPARIN THERAPY

Main adverse effects of heparin therapy include bleeding, thrombocytopenia and osteoporosis. Patients at particular risk are those who have had recent surgery or trauma or other clinical factors that predispose to bleeding like peptic ulcer, malignancy or liver disease.

Heparin should be discontinued temporarily or permanently. Protamine sulfate can be administered for urgent reversal.

Heparin induced thrombocytopenia, usually occurs in 1% to 2% of patients within 5 to 10 days (Kelton JG 1986) (36). 50% fall in platelet count occurs, due to immunoglobulin G directed against a complex of platelet factor 4 and heparin (Greinacher A et al 1991) (27).

Warkoentin TE et al (1997) reported that the development of thrombocytopenia may be accompanied by arterial or venous thrombosis which may lead to serious consequences such as death or limb amputation. Heparin in all forms must be stopped and treatment instituted with Danaproid, Hirudin or Argatroban.

Osteoporosis has been reported in patients receiving unfractionated heparin in dosages of 20,000 units/day for more than 6 months.

ORAL ANTICOAGULANT THERAPY

The anticoagulant effect of warfarin is mediated by the inhibition of the vitamin K dependent gamma carboxylation of coagulation factors II, VII, IX and X. This results in the synthesis of inactive coagulation proteins. It also inhibits protein C & S. Since these have a shorter half lives than the coagulation factors, warfarin causes a period of initial paradoxical thrombogenecity (Clouse LH et al 1986).

Heparin and warfarin treatment should overlap for 4 to 5 days. The anticoagulant effect of warfarin is delayed until the normal clotting factors are cleared and the peak effect does not occur until 36 to 72 hours.

The laboratory test used to measure the effects of warfarin is prothrombin time. The daily dose is adjusted to maintain International Normalized Ratio (INR) between 2 and 3 and heparin is discontinued. For patients experiencing a first episode of venous thromboembolism, long term anticoagulant therapy should be continued for atleast 3 to 6 months and for patients with recurrent event, anticoagulant should be continued for a longer time.

Brandjes DPM et al (1997) (7) showed that the use of graduated compression stockings for 24 months significantly decreased the incidence of post-thrombotic

syndrome. The major adverse effect of warfarin therapy is bleeding Levine MN et al (1989) (40).

Bleeding during well controlled oral anticoagulant therapy is usually due to trauma or local lesions such as peptic ulcer. Spontaneous bleeding can occur if excessive dosage of warfarin is given. Coumarin induced skin necrosis is a rare but serious complication that requires immediate cessation of oral anticoagulant therapy (Beeker CG 1987) (4). It usually occurs between 3 and 10 days after therapy, is commoner in women and most often involves areas of abundant subcutaneous tissue such as abdomen, buttock, thighs and breast.

VENOUS THROMBECTOMY

Mahorner's (4) pioneering work published in 1954 advocating thrombectomy showed there was dramatic reduction of leg edema, no pulmonary embolism and no late morbidity.

Initially surgeons avoided thrombectomy because of the 9-10% mortality.

To avoid perioperative pulmonary embolism, we must have a preoperative venogram to exclude thrombus extension into inferior vena cava and using intraoperative PEEP.

In Sweden Plate G et al (1985) (48) in a randomized study, found positive perfusion scans in 45% of all patients on admission with additional defects seen after 1 and 4 weeks in 11% and 12% of conservatively treated group and 20% and 0% in thrombectomized group respectively. In this series no additional perfusion defects developed after the first postoperative week following thrombectomy and arteriovenous

fistula. Since the arteriovenous fistula prevented rethrombosis, we can assume that the fistula was one reason for the low incidence of pulmonary embolism.

Plate G et al also reported only a 13% rethrombosis rate after arteriovenous fistula.

Post operative wound hematoma, wound infection and lymph leak are other complications.

Plate G et al (1990) (47) study also showed that at 5 years 37% of operated patients were asymptomatic compared with 18% in conservatively treated group. The figures were 54% and 23% at the end of 10 years.

Iliac vein patency too was 77% and 47% at the end of 10 years in the surgery and conservative group.

THROMBOLYSIS

The standard regimen of systemic anticoagulation with heparin followed by coumadin therapy does not promote lysis to reduce the thrombus load nor does it restore venous function. Thrombolysis is a potentially attractive form of therapy since it provides the opportunity for promptly restoring venous patency and preserving venous valve function.

In a pooled analysis of 13 randomized studies, Comerota and Alridge (11) found that only 4% of patients treated with heparin had significant or complete lysis compared to 45% of patients randomised to systemic lytic therapy. Jeffrey and Co-workers (33) in a 5 year follow up of those subjected to systemic streptokinase, showed that those who

achieved complete lysis had only a 9% incidence of popliteal reflux, whereas those with incomplete lysis had a 77% incidence of popliteal reflux.

The report by Semba and Dake in 1994 (54) provided the first insight on the potential role of catheter directed thrombolysis. They achieved 72% complete and 20% significant partial lysis in the 93% of cases in which successful access was achieved. There was 88% patency at 1 year.

Access was obtained most commonly through popliteal vein under ultrasound guidance.

CAVAL INTERRUPTION

In 1893, Bothini successfully performed ligation of the inferior vena cava.

Nasbeth DC et al (1965) (44) found mortality rates after inferior vena cava ligation to be 19% and leg edema 40%, varicose vein 20% stasis pigmentation (18%) venous claudication 14% and ulceration 6%. Hence Diccone VA et al (1970) (46) suggested plication of inferior vena cava instead of ligation.

IVC FILTERS

Intraluminal devices were subsequently developed and placed in the inferior vena cava by surgery (Mobin – Uddin Umbrellas) The Kimray – Greenfield stainless steel filter was introduced in 1973. The next step in the development of the filters was the transcutaneous placement of IVC filters. Filter placement through femoral vein was reported by Denny DF et al in 1985 (18).

But Alexander et al (1994) (1) reported a higher incidence of morbidity (pulmonary embolism, caval thrombosis, renal dysfunction, venous gangrene, insertion

site thrombosis, filter misplacement, migration and bleeding) when filters were inserted percutaneously compared to surgical placement. Insertion of the large size filters caused insertion site thrombosis and this lead to the development of small filters that could be loaded through much smaller introducer sheaths.

Becker and Colleagues (1992) (5) combined the results of 13 consecutive series of patients with pulmonary embolism who were treated with Greenfield IVC filters. They described on overall frequency of 2.4% of recurrent pulmonary embolism and 0.7% recurrent fatal pulmonary embolism.

Ballew et al (1995) (2) reviewed 11 series of patients with pulmonary embolism treated with filters and described an overall frequency of 2.9% of recurrent pulmonary embolism and a frequency of 0.8% recurrent fatal pulmonary embolism. Comparing these data with previous reports strongly suggest that IVC filters prevent fatal pulmonary embolism.

ANALYSIS AND DISCUSSION

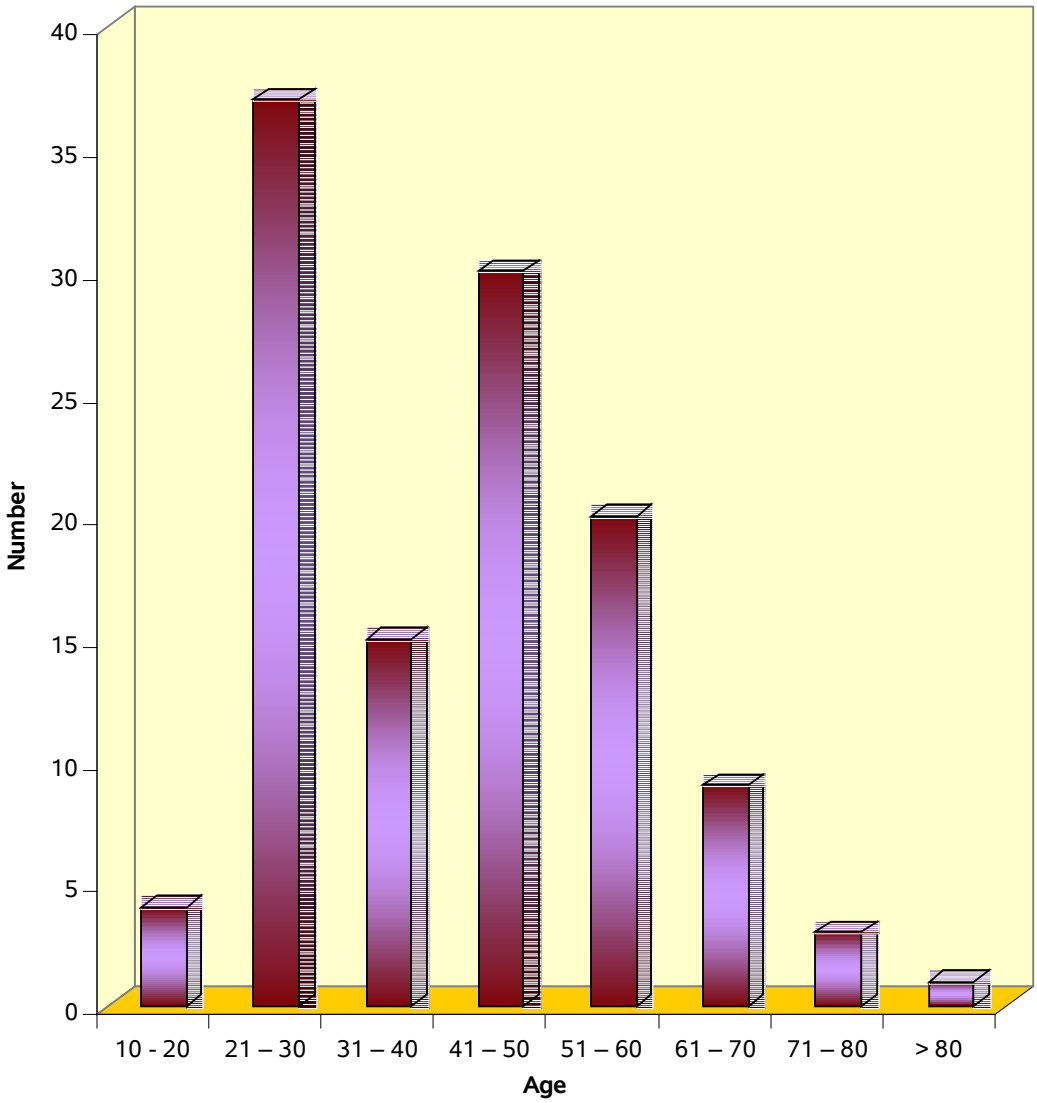
PRESENTATION

All the 119 patients presented with limb edema and pain. Out of this there were two cases of left subclavian and axillary vein thrombosis, the rest were lower limb deep vein thrombosis.

None of the patients presented with phlegmasia alba dolens or cerulea dolens.

All of them underwent a chest xray, ECG and echocardiographic examination and no detectable pulmonary embolism was present. Also there were no mortality.

AGE INCIDENCE



Out of 4 patients in the 10-20 year age group, three of them had a predisposing factor like surgery, burns and neurological condition. The remaining one had a recurrent episode and most probably suffers from a hypercoagulable state.

There were 37 patient in the 21-30 age group mostly because of pregnancy and puerperium related deep vein thrombosis.

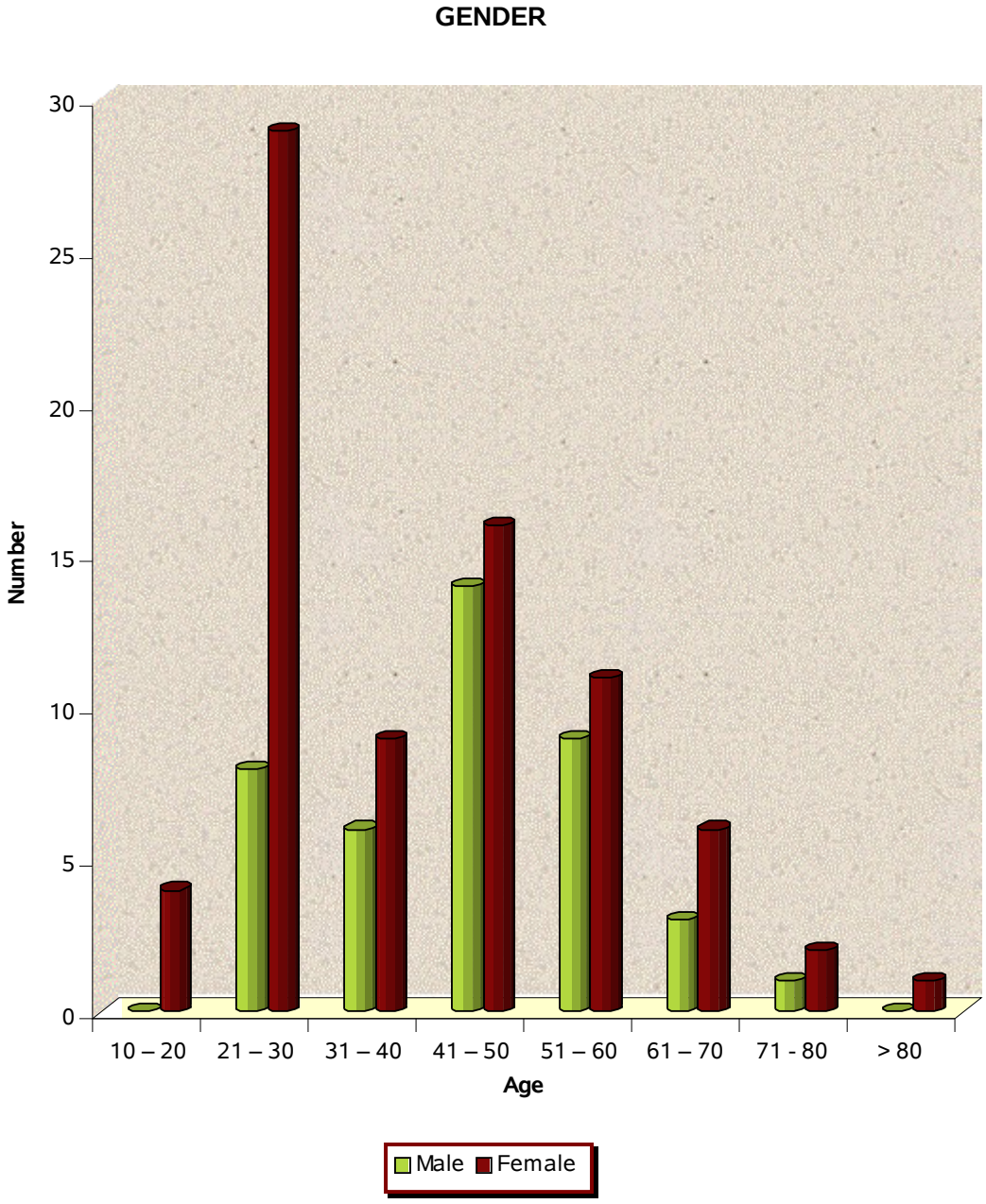
Almost 50% of patients fall within 41-60 years age group. The more common predisposing factors were trauma, surgery, idiopathic and tumor.

GENDER

Male 41

Female 78

Age	Male	Female	Total
10 – 20	-	4	4
21 – 30	8	29	37
31 – 40	6	9	15
41 – 50	14	16	30
51 – 60	9	11	20
61 – 70	3	6	9
71 – 80	1	2	3
> 80	-	1	1

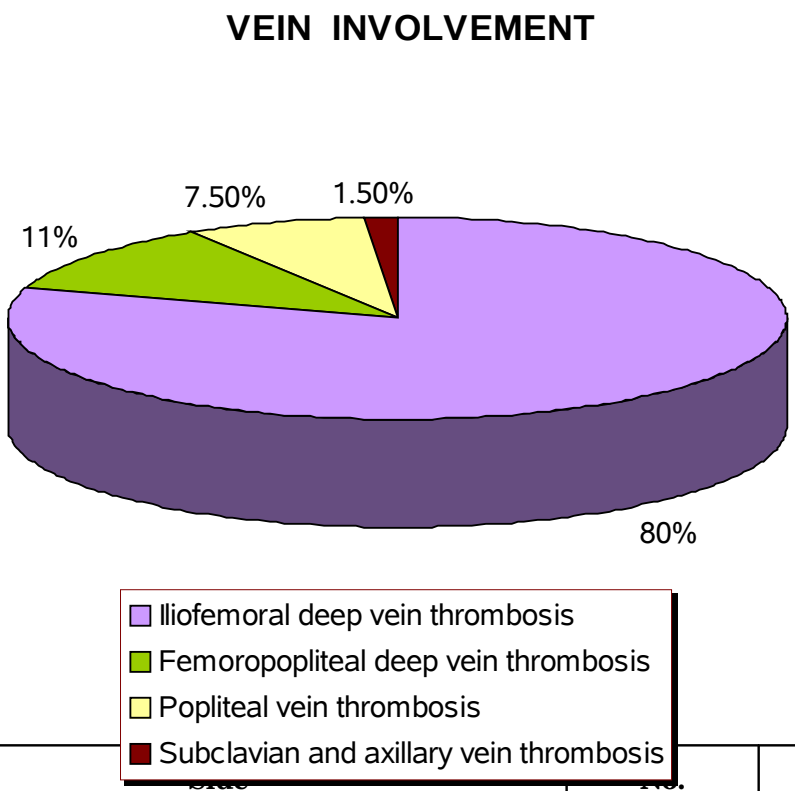


The risk imparted by gender remains uncertain. There is a higher incidence among females during child bearing years and equal between the sexes in the above 40 age group.

LIMB INVOLVEMENT

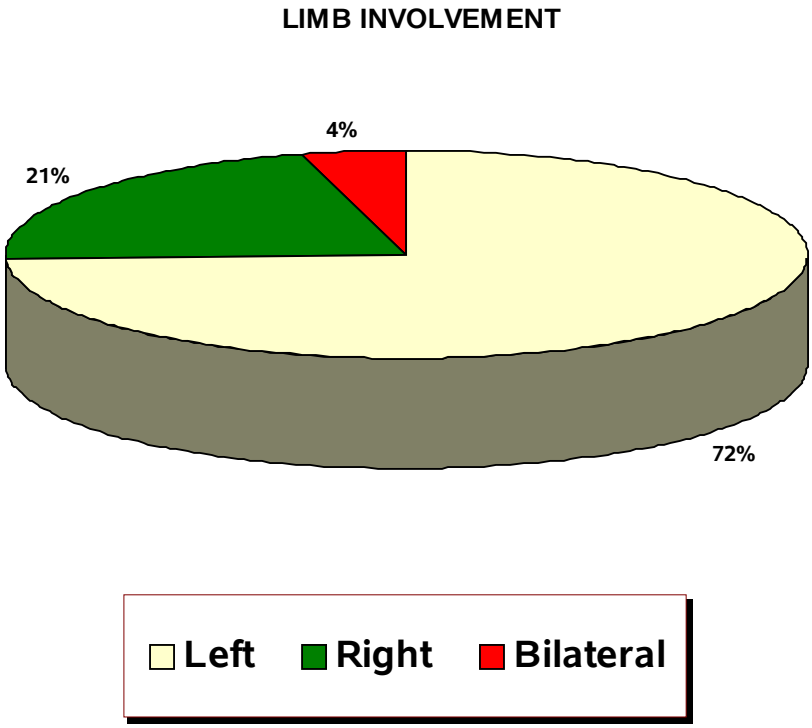
There were 117 cases of lower limb deep vein thrombosis and 2 upper limb venous thrombosis.

	No.	%
Iliofemoral deep vein thrombosis	95	80%
Femoropopliteal deep vein thrombosis	13	11%
Popliteal vein thrombosis	9	7.5%
Subclavian and axillary vein thrombosis	2	1.5%



	[illegible]	%
Left	89	72%

Right	25	21%
Bilateral	5	4%



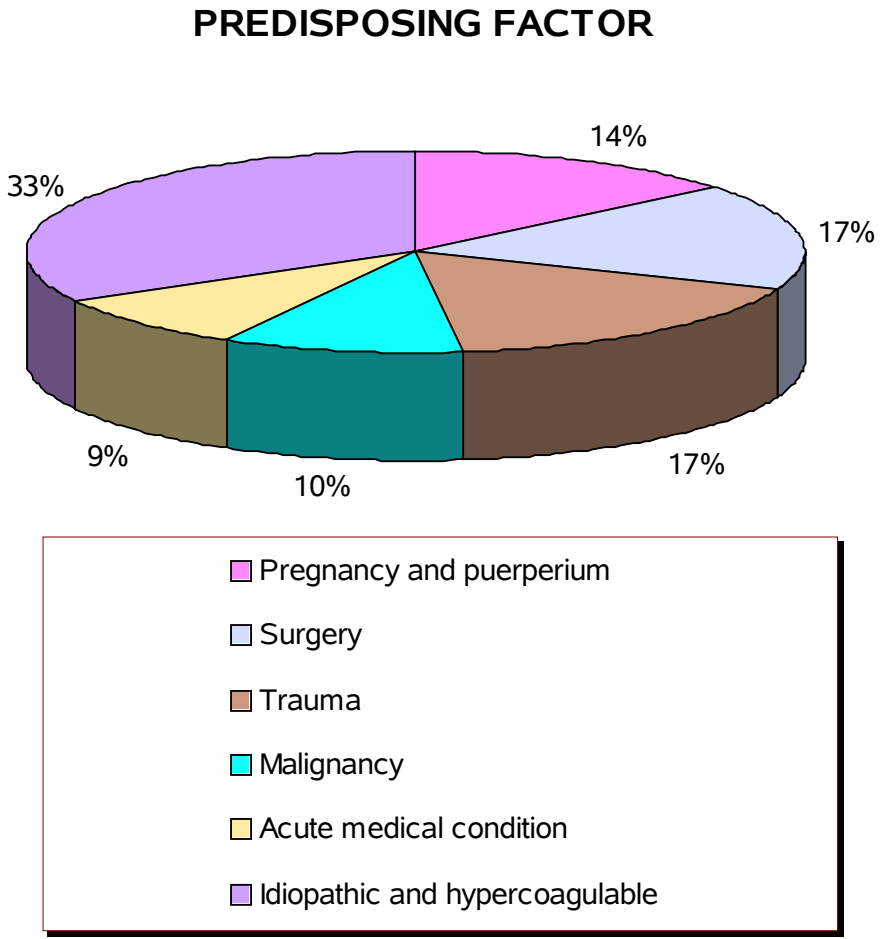
The number of patients presenting with iliofemoral venous thrombosis was 95 (79.83%). This is because most of the patients ignored their initial symptom of leg swelling and presented only when the whole of the lower limb upto the groin became swollen and developed pain.

There were 2 cases of left subclavian and axillary vein thrombosis. Both of them did not have any known predisposing factor like central venous catheter.

The number of left lower limb involvement was more (75%) when compared to right 21%. This correlates with book description. One of the reasons cited is the compression of the left common iliac vein by the right common iliac artery.

Chronic compression may cause structural damage to the vein and may be a reason for recurrent deep vein thrombosis. May Thurner described a syndrome where there are intimal webs in the left common iliac vein.

Predisposing factor	No.	%
Pregnancy and puerperium	18	14
Surgery	20	17
Trauma	21	17
Malignancy	12	10
Acute medical condition	11	9
Idiopathic and hypercoagulable	41	33
Total	**119**	**100**



PREGNANCY AND PUERPERIUM

Out of the 78 patients who developed deep vein thrombosis, 18 of them were either pregnant or postpartum state. 3 patients were in antepartum period, 9 of them developed following a normal delivery and 6 of them following a caesarean section.

During pregnancy the gravid uterus compresses the inferior vena cava and iliac veins causing sluggish venous flow and this added with surgical intervention and immobilisation has increased the deep vein thrombosis risk.

POST SURGERY : 17%

The highest risk for venous thromboembolism is imparted by hospitalisation for surgery.

Total no. of patients with deep vein thrombosis following surgery	:	20	
General surgical procedure	:	4	
Orthopaedic	:		3
Neurosurgical	:	2	
Cancer surgery	:		5
Gynaecological	:	6	

This excludes the post caesarean section patients. Patients who undergo prolonged surgical procedures are at risk of developing venous thromboembolism. The risk continues even after discharge from hospital. In this study 5 out of the 6 gynaecologic surgery patients developed limb edema after the 10^{th} postoperative day after being discharged.

The orthopaedic and neurosurgical patients developed deep vein thrombosis in their 2nd and 3rd postoperative days. The orthopaedic procedure was a hip arthroplasty and a cervical discectomy.

MALIGNANCY : 10%

Out of the 119 cases, 12 patients had malignant lesions.

Ca Cervix	4
Ca Rectum	3
Ca Pancreas	1
Ca Stomach	1
Ca Bladder	1
Renal cell carcinoma	1
Ca Breast	1

Al the carcinoma cervix patients had underwent prior radiotherapy either external or interstitial 6 other cancer patients had underwent surgery and developed deep vein thrombosis in the postoperative period.

TRAUMA : 17%

Post traumatic deep vein thrombosis constitutes about 17% of the total number of deep vein thrombosis. Out of the 21 patients 16 had either fracture or head injury.

Extremity bone fracture	4
Spine fracture	9
Pelvic fracture	1
Head injury	2
Femoral vein cannula	5
Total	**21**

All the 4 patients with fracture of long bones developed ipsilateral deep vein thrombosis. Patients with fracture spine either had paraplegia or quadriplegia.

There were 2 cases of head injury, with subarachnoid hemorrhage and one patient with pelvic fracture.

Trauma along with immobilisation were supposed to be the predisposing causes in these patients.

There is a different group of patients who developed deep vein thrombosis following femoral vein cannulation. Four of them were heart block patients who had temporary pace maker insertion and one patient had cannula for hemodialysis.

ACUTE MEDICAL CONDITION (9%)

Eleven patients who had acute medical condition and were undergoing intensive care treatment developed deep vein thrombosis (9%). This compares well with international data. The importance of thromboembolic disease arising in the medical

wards is confirmed by the fact that three quarters of patients presenting with pulmonary embolism are non surgical.

IDIOPATHIC AND HYPERCOAGULABLE STATES (33%)

This constitutes the largest group of patients in this study. Patients in whom there were no obvious predisposing factors were termed idiopathic group. Few patient who could afford the thrombophilia screening were investigated accordingly. Inherited thrombophilia is a genetically determined tendency to deep venous thrombosis. The true prevalence of inherited thrombophilia in the general population is presently unknown, since we do not know all genetic abnormalities causing a tendency to venous thrombosis. Indirect evidence suggests, that it might be much higher than estimates from prevalence studies on the known genetic defects. Hence we have combined the idiopathic and hypercoagulable groups into one. Two patients had protein C deficiency, 2 had elevated antiphospholipid IgG antibodies and one patient had hyper homocysteinemia. Interestingly the last patient, a 29 year old male also suffered from severe coronary artery disease.

One patient with antiphospholipid antibody had an episode of cortical vein thrombosis 6 months prior to the lower limb venous thrombosis and the other had 2 abortions.

There were also 6 cases of recurrent episodes. These patients have discontinued their anticoagulants without medical advice.

SUMMARY

- In this study all patients presented only with limb edema and pain. No case of phlegmasia albo dolens or cerulea dolens was seen.
- No fatal pulmonary embolism was encountered.
- The left lower limb veins were involved in almost 75% of patients.
- Iliofemoral venous thrombosis constituted 80% of the total cases studied.
- Surgery and trauma together constituted the most common causes predisposing to deep vein thrombosis and which can be prevented.
- Idiopathic group constituted the majority of patients. This group needs further evaluation to rule out the presence of inherited thrombophilias.
- In uncomplicated lower limb venous thrombosis, conservative treatment with heparin and oral anticoagulants seems to produce acceptable results in this set up.

CONCLUSION

- The myth that Asians are immune to deep vein thrombosis has been dispelled.
- Appropriate prophylaxis in high risk patients needs to be instituted.
- Screening of patients with thromboembolic events irrespective of the modality of presentation has the potential to identify patients requiring a prolonged course of anticoagulation.

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67.

PROFORMA

Name	:		
Age	:		
Sex	:		
IP No.	:		
Date of Admission	:		
Symptoms	Edema	Pain	Others
Site of DVT	Iliofemoral		Unilateral
	Femoropopliteal		Bilateral
	Popliteal		
	Others		
Duplex Scan	:		
Symptoms of Pulmonary Embolism	:	Yes	No
Previous DVT	:		
Thrombophilia Screening done	:	Yes	No
Presence of			
Varicose veins	:		
Cancer	:		
Long distance travel	:		

If female

Ocpill usage :

HRT :

Pregnancy :

Postpartum :

Acute medical condition within previous 2 months : Yes No

Major surgery within past 2 months : Yes No

Major trauma / fracture within past 2 months : Yes No

DVT prophylaxis : Yes No

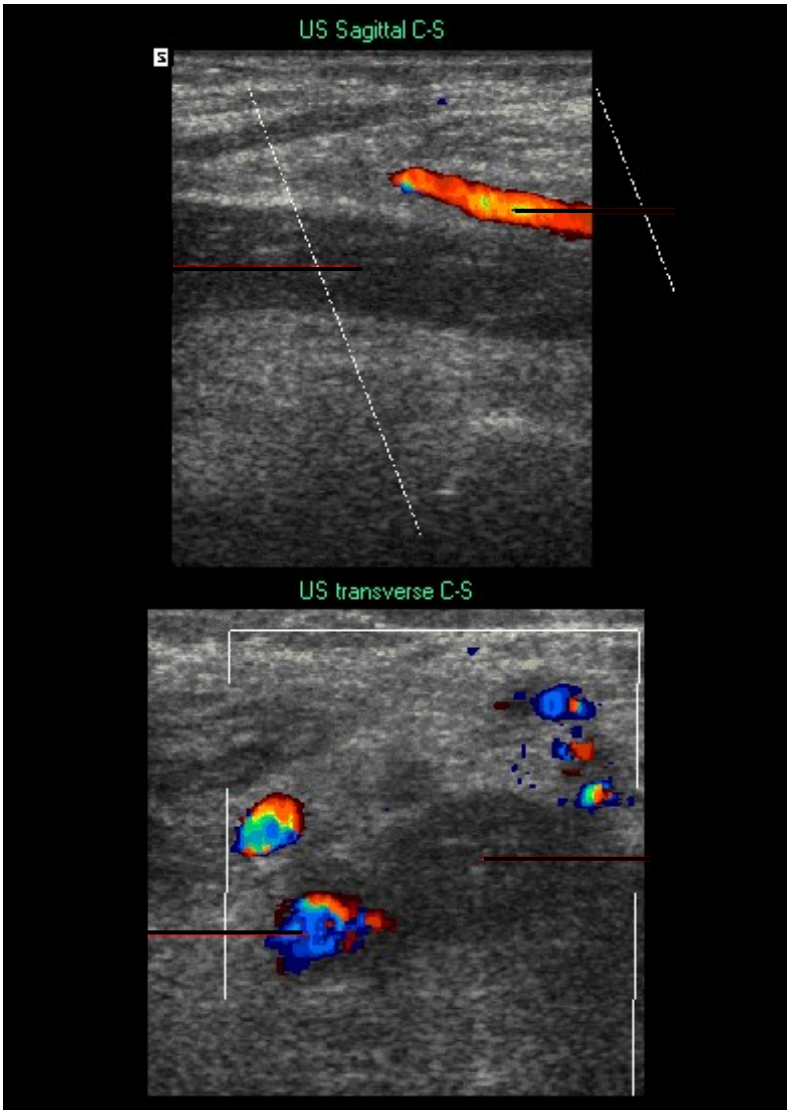
Treatment

Heparin :

Acitrom :

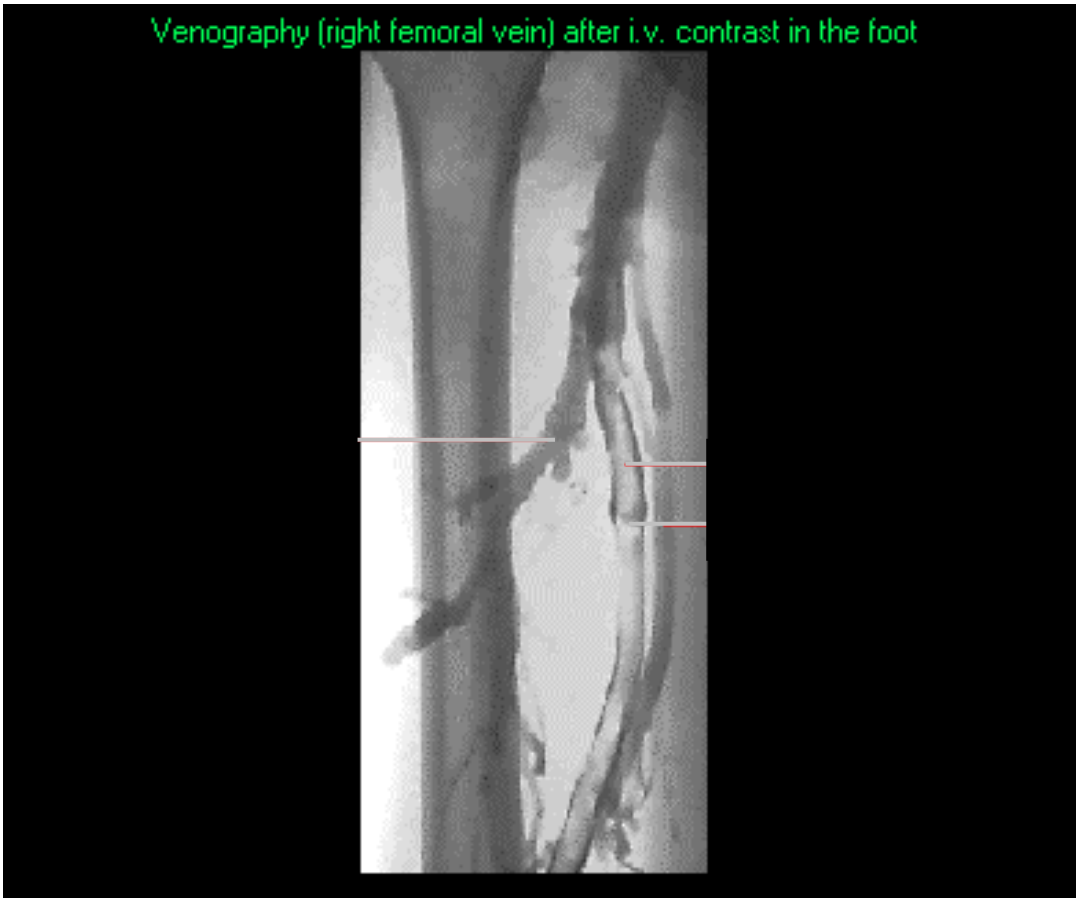
Compression stockings :

DUPLEX SCAN SHOWING POPLITEAL VEIN THROMBUS

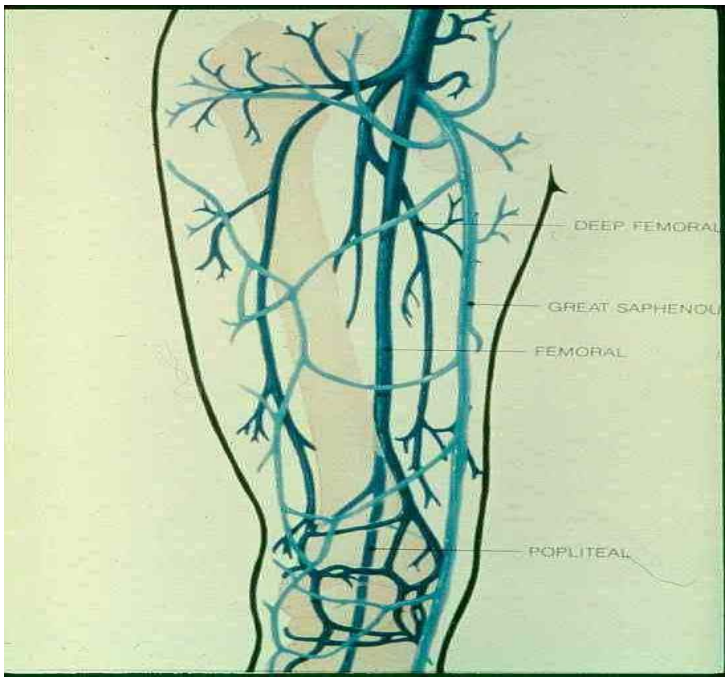


VENOGRAM SHOWING FEMORAL

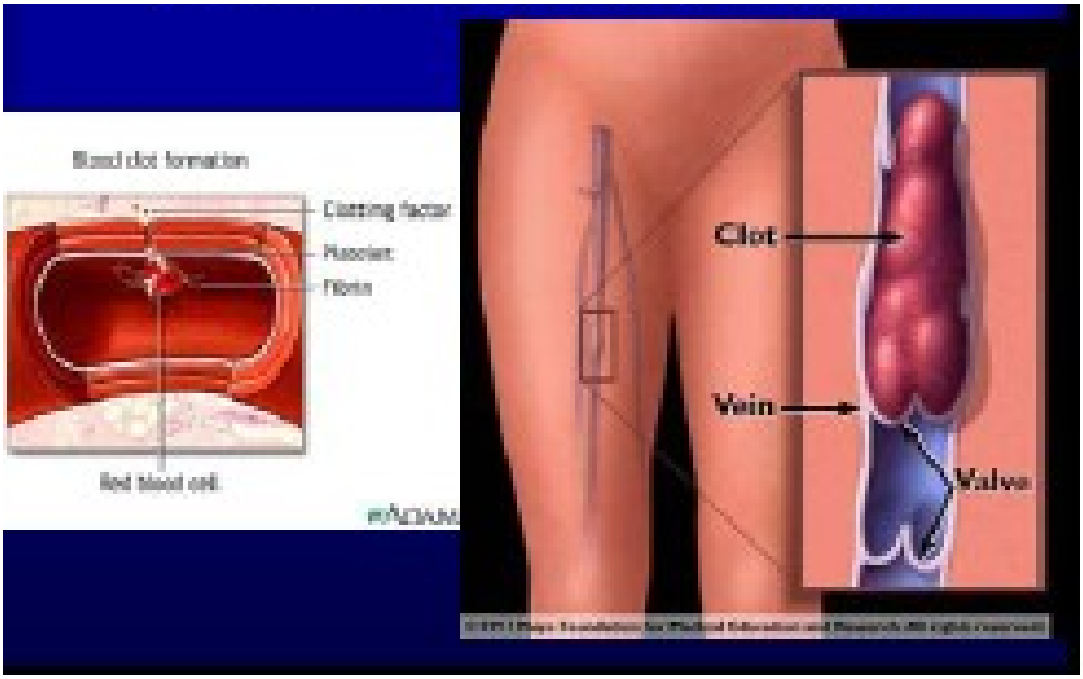
VEIN THROMBUS



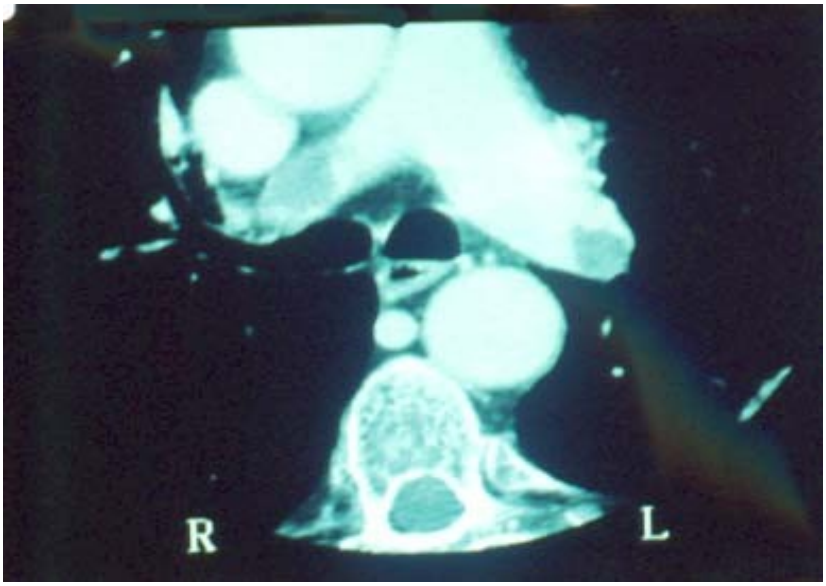
ANATOMY OF LOWER LIMB VEINS



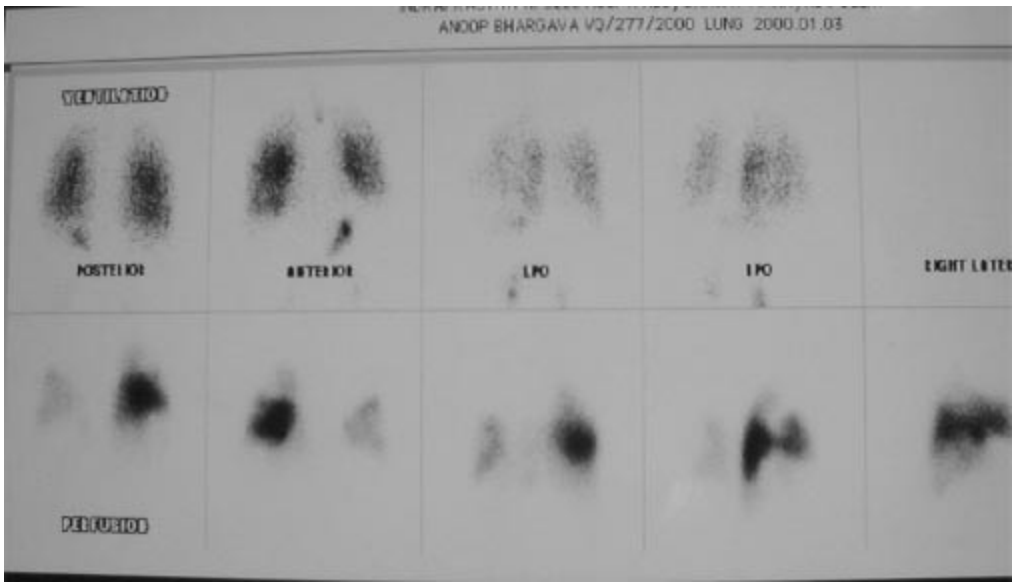
PATHOLOGY OF DVT



A SPIRAL CT ANGIOGRAM SHOWING PROXIMAL PULMONARY EMBOLI



VENTILATION – PERFUSION SCAN



High Probability Ventilation Perfusion Scan images showing mismatched segmental perfusion defects.

ACUTE LEFT ILIOFEMORAL DVT

POST PARTUM DVT

DVT WITH BLEBS

WITH COMPRESSION BANDAGE

POST PHLEBITIC LEG

.No.	Name	Age	Sex	No	DOA	Site	Predisposing
1	Yasodammal	45	F	668910	05.07.04	(L) IFDVT	Idiopathic
2.	Rahman	56	M	669772	09.07.04	(L) IFDVT	Idiopathic
3.	Poongavanam	50	F	669761	09.07.04	(L) IFDVT	Ca cervix
4.	Nagammal	30	F	669869	09.07.04	(L) IFDVT	Idiopathic
5.	Chari	50	M	670333	12.07.04	(L) IFDVT	Idiopathic
6.	Panchanathan	38	M	671141	16.07.04	BIL IFDVT	Idiopathic
7.	Kanagambaran	41	F	672856	26.07.04	(L) IFDVT	Post OP
8.	Banumathi	22	F	672890	26.07.04	(L) IFDVT	Post LSCS
9.	Lakshmi	25	F	673601	30.07.04	(L) FPDVT	Post partum
10.	Kanniyammal	53	F	666081	20.07.04	(L) IFDVT	Trauma
11.	Hamsa	25	F	171821	16.07.04	(L) IFDVT	Post LSCS
12.	Thilagam	19	F	674875	06.08.04	(L) IFDVT	Post LSCS
13.	Gnanamani	70	F	675303	09.08.04	(L) IFDVT	Idiopathic
14.	Peeramana	50	F	675306	09.08.04	BIL IFDVT	Ca. Pancreas
15.	Muniamma	48	F	675347	09.08.04	(L) IFDVT	Idiopathic
16.	Geetha	40	F	675402	01.08.04	(R) FPDVT	Post OP
17.	Malla	45	F	673850	02.08.04	(R) IFDVT	Trauma
18.	Savithiri	41	F	676368	15.08.04	(R) IFDVT	Idiopathic
19.	Lakshmiammal	50	F	678490	26.08.04	(L) IFDVT	Idiopathic
20.	Moses	45	M	678904	19.08.04	(R) POP DVT	Trauma
21.	Hameed	50	M	678453	24.08.04	(R) IFDVT	Cannula
22.	Radhi	25	F	679574	02.09.04	(L) IFDVT	Post LSCS
23.	Jeyalaxmi	46	F	680318	06.09.04	(L) IFDVT	POST OP
24.	Lakshmi	55	F	678835	20.08.04	(L) FPDVT	Trauma
25.	Pavalakodi	55	F	679876	20.08.04	(L) IFDVT	Trauma
26.	Ayyadurai	40	M	680941	01.09.04	(L) SCDVT	Acute Med. Cond.
27.	Sonabee	65	F	680104	01.09.04	(L) FPDVT	Trauma
28.	Satamthala	63	F	680987	09.09.04	(L) IFDVT	Idiopathic
29.	Pachaiyappan	49	F	681560	13.09.04	(L) IFDVT	Idiopathic
30.	Shanthi	28	F	682089	16.09.04	(L) IFDVT	Post partum

.No.	Name	Age	Sex	No	DOA	Site	Predisposing
31.	Thangavel	56	M	683530	24.09.04	(R) IFDVT	Idiopathic
32.	Malarkodi	22	F	683732	27.09.04	BIL IFDVT	Pregnancy
33.	Natraj	41	M	684568	30.09.04	(L) IFDVT	Post OP
34.	Perumal	65	M	685096	04.10.04	(R) FPDVT	Acute Med. Cond.
35.	Rena	44	F	685782	07.10.04	(L) IFDVT	Idiopathic
36.	Alamelu	47	F	685740	07.10.04	(L) IFDVT	Post OP
37.	Sasikala	21	F	685851	10.10.04	(L) IFDVT	Post partum
38.	Amuda	50	F	685536	05.10.04	(L) IFDVT	Acute Med. Cond.
39.	Nallayi	57	F	685018	01.10.04	(R) IFDVT	Cannula
40.	Shankar	35	M	685103	01.10.04	(L) IFDVT	Acute Med.Cond.
41.	Jayagandhi	24	F	687731	18.10.04	(L) IFDVT	Post partum
42.	Chandra	50	F	687440	16.10.04	(L) IFDVT	Ca.Stomach
43.	Umabanu	26	F	689403	28.10.04	(L) IFDVT	Idiopathic
44.	Anusuya	55	F	689467	29.10.04	(L) IFDVT	Post OP
45.	Ellammal	57	F	691779	13.10.04	(L) IFDVT	Ca Cervix
46.	Subramani	47	M	689753	19.11.04	(R) POP DVT	Cannula
47.	Chitra	25	F	690132	22.11.04	(L) FPDVT	Trauma
48.	Gandhimathi	26	F	694963	30.11.04	(L) IFDVT	Pregnancy
49.	Perumal	55	M	690741	05.11.04	(L) IFDVT	Idiopathic
50.	Badrunnisha	85	M	695803	11.12.04	(L) IFDVT	Idiopathic
51.	Jayalakshmi	50	F	689885	19.12.04	(L) IFDVT	Ca breast
52.	Vijayalakhsmi	35	F	689710	10.12.04	BIL IFDVT	Ca rectum
53.	Saraswathy	64	F	689690	03.12.04	(L) IFDVT	Acute Med. Cond.
54.	Kumar	43	M	700053	27.12.04	(L) POPDVT	Idiopathic
55.	Ahamed Rasheed	21	M	698632	21.12.04	(R) IFDVT	Idiopathic
56.	Ramanya	45	M	697814	16.12.04	(R) IFDVT	Idiopathic
57.	Krishnan	64	M	700478	30.12.04	(L) FPDVT	Idiopathic
58.	Kuppammal	73	F	701560	06.01.05	(L) IFDVT	Trauma
59.	Mohan	50	M	701440	05.01.05	(L) POPDVT	Trauma
60.	Rajendran	55	M	697425	05.01.05	(R) POPDVT	Post OP

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61.	Mariya Stephen	54	M	702809	29.01.05	(L) IFDVT	Trauma
62.	Selvaraj	55	M	706997	07.02.05	(L) IFDVT	Idiopathic
63.	Thilagavathy	27	F	707523	10.02.05	(L) IFDVT	Idiopathic
64.	Nirmala	37	F	707624	11.02.05	(L) IFDVT	Pregnancy
65.	Pushpa	30	F	707671	11.02.05	(R) IFDVT	Post partum
66.	Subramani	22	M	706579	06.02.05	(L) POPDVT	Trauma
67.	Govindammal	52	F	700259	06.02.05	(R) FPDVT	Cannula
68.	Ashya Parveen	22	F	711778	01.03.05	(L) IFDVT	Post LSCS
69.	Kumari	15	F	712748	07.03.05	(L) FPDVT	Idiopathic
70.	Mani	50	M	713379	10.03.05	(R) IFDVT	Idiopathic
71.	Rohini	55	F	713048	10.03.05	(R) IFDVT	Idiopathic
72.	Kumar	69	M	834105	10.03.05	(R) IFDVT	Trauma
73.	Abdul Saleem	45	M	697838	11.03.05	(L) IFDVT	Post OP
74.	Krishnaveni	45	F	661972	25.03.05	(L) IFDVT	Post OP
75.	Nirmala	25	F	714057	15.03.05	(L) IFDVT	Post partum
76.	Lourdemary	35	F	715322	21.03.05	(L) IFDVT	Acute Med. Cond.
77.	Yasoda	65	F	716292	27.03.05	(L) IFDVT	Post OP
78.	Sandya	24	F	717132	31.03.05	(L) IFDVT	Idiopathic
79.	Thangavel	50	M	717635	31.03.05	(R) IFDVT	Idiopathic
80.	Gopi	55	M	720067	18.04.05	(L) IFDVT	Idiopathic
81.	Probudoss	28	M	720097	18.04.05	(L) IFDVT	Idiopathic
82.	Chinnadurai	50	M	714311	02.04.05	(L) IFDVT	Trauma
83.	Indra	50	F	720118	04.04.05	(R) FPDVT	Idiopathic
84.	Elizabeth	48	F	721018	23.04.05	(L) IFDVT	Idiopathic
85.	Pushpavathy	75	F	720748	19.04.05	(R) IFDVT	Ca Cervix
86.	Padmavathy	36	F	722042	28.04.05	(L) IFDVT	Post OP
87.	Nirmala	24	F	721582	26.04.05	(L) IFDVT	Post LSCS
88.	Kasthuri	37	F	724010	09.05.05	(L) IFDVT	Idiopathic
89.	Mahalingam	44	M	726216	16.05.05	(L) IFDVT	Post OP
90.	Vasu	29	M	727645	23.05.05	(L) POPDVT	Idiopathic

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91.	Saroja	58	F	727643	23.05.05	(R) IFDVT	Ca Cervix
92.	Ramani	27	F	727699	24.05.05	(L) IFDVT	Idiopathic
93.	Thangavel	40	M	727907	26.05.05	(L) IFDVT	Acute Med.Cond.
94.	Chandrasekar	28	M	715276	29.05.05	(L) POPDVT	Ca Rectum
95.	Rajammal	60	F	729286	31.05.05	(L) IFDVT	Idiopathic
96.	Revathy	10	F	717835	17.05.01	(L) IFDVT	Acute Med. Cond.
97.	Angammal	72	F	721636	10.05.05	(L) IFDVT	Trauma
98.	Bebya	25	F	726618	19.05.05	(R) IFDVT	Acute Med. Cond.
99.	Giri	30	M	729659	02.06.05	(L) AXIDVT	Idiopathic
100.	Babu	40	M	730936	14.06.05	(L) IFDVT	Renal Cell Ca
101.	Sanmyammal	45	F	729496	20.06.05	(L) IFDVT	Trauma
102.	Balasundaran	45	F	729496	20.06.05	(L) IFDVT	Trauma
103.	Venkatesh	23	M	734342	29.06.05	(L) POPDVT	Post OP
104.	Jecintha	22	F	731005	09.06.05	(L) IFDVT	Post OP
105.	Pitchai Mohd	53	M	735089	30.06.05	(R) IFDVT	Idiopathic
106.	Anjalai	27	F	739371	19.07.05	(L) IFDVT	Idiopathic
107.	Radabai	55	F	735593	13.07.05	(L) IFDVT	Trauma
108.	Mallika	40	F	736010	05.07.05	(L) IFDVT	Ca Rectum
109.	Varalakshmi	22	F	738402	14.07.05	BIL.IFDVT	Post partum
110.	Kotteswaran	34	M	736635	19.07.05	(L) IFDVT	Trauma
111.	Ruckmani	65	F	737614	13.07.05	(R) FPDVT	Cannula
112.	Balakrishnan	54	M	734098	19.07.05	(R) FPDVT	Ca bladder
113.	Malar	17	F	811005	08.07.05	(L) IFDVT	Post OP
114.	Kala	24	F	738200	14.07.05	(L) IFDVT	Idiopathic
115.	Soundari	25	F	739684	22.07.05	(L) IFDVT	Post partum
116.	Ramalingam	30	M	739772	21.07.05	(L) IFDVT	Idiopathic
117.	Kasthrui	24	F	736579	01.07.05	(L) IFDVT	Post partum
118.	Kavitha	25	F	741305	28.07.05	(L) IFDVT	Idiopathic
119.	Sivagami	37	F	740997	27.07.05	(L) IFDVT	Idiopathic

IFDVT	-	Iliofemoral DVT
FPDVT	-	Femoropopliteal DVT
POPDVT	-	Popliteal DVT
AXIDVT	-	Axillary DVT
SCDVT	-	Subclavian DVT